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Abstract

The reactivity hypothesis postulates that large magnitude cardiovascular reactions to psychological stress contribute to the development of pathology. A key but little tested assumption is that such reactions are metabolically exaggerated. Cardiac activity, using Doppler echocardiography, and oxygen consumption, using mass spectrometry, were measured at rest and during and after a mental stress task and during graded sub maximal cycling exercise. Cardiac activity and oxygen consumption showed the expected orderly association during exercise. However, during stress, large increases in cardiac activity were observed in the context of modest rises in energy expenditure; observed cardiac activity during stress substantially exceeded that predicted on the basis of contemporary levels of oxygen consumption. Thus, psychological stress can provoke increases in cardiac activity difficult to account for in terms of the metabolic demands of the stress task.

Descriptors: Additional cardiac activity, Cardiac output, Exercise, Heart rate, Oxygen consumption, Psychological stress

The reactivity hypothesis postulates that large magnitude cardiovascular reactions to psychological stress contribute to the aetiology of cardiovascular pathology (Lovallo & Gerin, 2003; Schwartz et al., 2003) and several prospective studies have now shown with reasonable consistency that high reactivity confers a modest additional risk for a range of cardiovascular disease outcomes, such as high blood pressure, carotid atherosclerosis, carotid intima-thickness, and increased left ventricular mass (e.g., Allen, Matthews, & Sherman, 1997; Barnett, Spence, Manuck, & Jennings, 1997; Carroll, Ring, Hunt, Ford, & Macintyre,2003; Kamarck et al., 1997; Lynch, Everson, Kaplan, Salonen,& Salonen, 1998; Markovitz, Raczynski, Wallace, Chettur, & Chesney, 1998; Treiber et al., 2003). However, there is a seeming paradox; why should cardiovascular perturbations to psychological and behavioral stress lead to pathology whereas broadly similar adjustments during physical exercise are rightly regarded as health protective and beneficial? One major possible difference, alluded to by the early proponents of the reactivity hypothesis, is that the latter are metabolically appropriate whereas the former may not be (Obrist, 1981). There is a fair amount of evidence from human psychophysiological studies suggesting that the cardiovascular, especially cardiac, reactions to psychological stress may indeed be exaggerated relative to what would be expected on the basis of contemporary levels of energy expenditure. The earliest evidence emerged from two field studies, which set the methodological tone for future laboratory studies. Heart rate and oxygen consumption were recorded simultaneously in helicopter and transport aircraft pilots during difficult flight maneuvers (Blix, Stromme, & Ursin, 1974) and in novice parachutists just prior to jumping (Stromme, Wikeby, Blix, & Ursin, 1978). In both studies, cardiac and metabolic activity were also monitored during dynamic exercise. Heart rate increased to these naturalistic stress exposures more than would be expected from contemporary oxygen consumption levels and the relationship evident between cardiac activity and energy expenditure during exercise. The authors characterized such excess as ''additional heart rate.'' Between the early 1980s and the early 1990s, there was a small flurry of laboratory studies, from our group and from Paul Obrist's group at Chapel Hill, suggesting that ''additional heart rate'' (e.g., Carroll, Turner, & Hellawell, 1986; Carroll, Turner, & Prasad, 1986; Langer et al., 1985; Turner & Carroll, 1985), as well as ''additional cardiac output'' (e.g., Carroll, Harris, & Cross, 1991; Sherwood, Allen, Obrist, & Langer, 1986) reactions were provoked by a variety of psychological stress tasks, including mental arithmetic, video games, and aversive reaction time avoidance tasks.

Although the reactivity hypothesis has continued to inspire literally hundreds of studies each year, the issue of the metabolic appropriateness of the cardiovascular reactions being elicited in these studies has fallen into neglect. It may be that more contemporary researchers regard the matter was settled. Such presumption may be a little premature. Even at the time, there were skeptics, who continued to champion the view that, for the most part, ''behaviourally-related heart rate variance can be accounted for by metabolic variation'' (Brener, 1987, p. 449). We believe that, given the seemingly unremitting popularity of the reactivity hypothesis, it is timely to revisit the issue of

whether laboratory stress tasks do indeed elicit metabolically exaggerated cardiac adjustments. A number of considerations rekindled our interest. First, the presumption that the cardiac adjustments to

acute psychological stress are metabolically exaggerated lies at the very heart of the reactivity hypothesis; if it proves to be untrue, the hypothesis almost certainly falls. Second, in the earlier studies, the main outcome was either heart rate, an imperfect proxy for cardiac output and contractility, or cardiac output, derived indirectly from impedance cardiography. Systematic replication using an alternative approach to measuring cardiac output would seem important, and in Doppler echocardiography we have a more direct, yet still non invasive, means of determining cardiac output. Third, over the years our knowledge of the optimally challenging components of psychological stress tasks has allowed us to develop much more provocative exposures (see, e.g., Veldhuijzen van Zanten et al., 2004). The question arises as to whether these more powerful stress tasks provoke greater cardiovascular reactions because they require more metabolic commitment or are they, indeed, eliciting even higher levels of additional cardiac activity than those that were apparent in the early studies?

The present study, then, revisited the issue of whether acute psychological stress exposures are associated with exaggerated cardiac activity using the established design in which cardiac and metabolic activity, indexed by oxygen consumption, were monitored at rest, during acute psychological stress, and subsequently during graded sub maximal dynamic exercise. Cardiac activity was measured by Doppler echocardiography and oxygen concentration in inspired and expired air by mass spectrometry. The stress task was one that we know provokes substantial cardiovascular reactions (e.g., de Boer et al., 2007). It was hypothesized that psychological stress would elicit increases in cardiac output and heart rate that were metabolically inappropriate.

Method

Participants

Twenty-four healthy male undergraduates, with a mean age of 21 (SD51.02) years, provided informed consent and participated in the study. Women were excluded due to the possibility of variations in reactivity with menstrual cycle phase (Hastrup & Light, 1984; Stoney, Matthews, McDonald, & Johnson, 1988) and the sensitivities involved in thoracic scanning. None of the participants smoked, had a history of a cardio-respiratory disease, a current illness or infection, or were taking prescribed medication. They were required to refrain from consuming food or caffeine within 2 h and from alcohol intake and vigorous exercise within 12 h of the testing session. The study was approved by the relevant Research Ethics Committee.

Design

This was a multicondition within-subject study. Briefly, following instrumentation and instruction, participants were allowed approximately 20 min to adapt to the laboratory environment. This was followed by an 8-min formal resting baseline, an 8-min psychological stress task, and a 4-min resting recovery period. Finally, another 4-min resting baseline period was followed by four 4-min bouts of cycle exercise of incrementally increasing demand.

Stress and Graded Submaximal Exercise Tasks

The psychological stress task was the paced auditory serial addition test (PASAT; Gronwall, 1977; Ring et al., 1999), which demonstrates good test–retest reliability (Willemsen et al., 1998). Briefly, participants were presented, via audio CD, a series of single digit numbers and required, in each case, to add any given number to the number presented next. They indicated their answer by depressing the appropriate number (2–18) on a locally fabricated key pad, and this answer was then relayed to a computer screen in view of both participant and experimenter. This version of the PASAT consisted of four consecutive 2-min periods of 50, 65, 75, and 100 digits at presentation rates of 2.4, 2.0, 1.6,

and 1.2 s, respectively. An experimenter stood 1 m distant from and adjacent to the participants and ostentatiously scored their answers. The task also involved elements of competition, reward, and punishment. A leader board was displayed prominently and participants informed that they should attempt to beat the five scores on the board. They were awarded 1000 points at the start of the task but lost 5 points for every addition they failed to compute correctly. The final points total served as the performance score. Finally, they received a brief burst of loud, aversive noise once during the first 5 of every 10 trials, coincident with an error where one was made or at the end of the series of five if no errors were made. This ensured that each participant received the same number of noise bursts. The addition of the loud noise component has been found to increase the effects of the PASAT on cardiovascular activity (Veldhuijzen van Zanten et al., 2004). The mean performance score was 835 (SD592.34). Throughout the four 4-min continuous bouts of submaximal aerobic exercise, participants pedalled at a constant 50 revolutions per minute, using the tachometer provided. For the first 4 min, there was no friction load on the wheel, but for the subsequent three exercise bouts increasing friction loads were applied to yield exercise power demands of 30, 60, and 90 W, respectively. All participants were able to comply with these requirements.

Apparatus and Procedure

The study was conducted in a temperature-controlled (20°C ± 2°C) laboratory. Throughout the session, participants semi-reclined on a couch specifically designed for exercise echocardiography (Ergoselect 1000L, Ergoline GmbH, Bitz, Germany). The couch had an integrated cycle ergometer and the facility to tilt laterally to allow participants' hearts to be imaged. Participants remained tilted to the left for all measurements. Echocardiographic measurements were performed using a Philips Sonos 7500 ultrasound machine with an S3 two-dimensional transducer (1–3 MHz). Digital images of spectral waveforms were recorded continuously for later analysis. For each measurement point, averages were obtained from three or more spectral waveforms recorded at the end of expiration or as close as possible to it. Using this approach, measurements for aortic blood flow could be averaged across 60-s intervals. The electrocardiogram and a respiratory waveform were also recorded. An apical five-chamber view of the heart was used with Doppler mode to identify flow through the aortic valve during systole. Using pulsed-wave spectral mode at a screen sweep speed of 100 mm·s^{-1}, the velocity profile of the aortic flow was obtained. Exaggerated cardiac reactivity 271Doppler sampling of the flow was taken immediately below the orifice of the aortic valve. The flow was quantified automatically using the velocity time integral, which is the mean distance through which blood travels in the outflow tract during ventricular contraction. Each measurement of velocity time integral was made from at least three velocity profiles taken toward the end of expiration. The diameter of the aortic valve was measured from a parasternal long axis view and the aortic valve area was calculated. Stroke volume was calculated from velocity time integral × the aortic valve area; cardiac output was calculated as heart rate × stroke volume. Metabolic rate, indexed by oxygen consumption, was measured on a breath-by-breath basis by assessing ventilation and analyzing inspired and expired gas composition using an integrated system. The apparatus used to supply air and measure ventilation was the mouthpiece assembly. Its key components were a turbine (VMM 400, Interface Associates, Laguna Niguel, CA, USA) to measure the volume of inspired and expired gas and a pneumotachograph (Hans Rudolf, Kansas City, MO, USA) to determine flow and respiratory timings. Gas composition was analyzed by a fast responding quadrupole mass spectrometer (Airspec, QP2000, Airspec, Kent, UK). All data were recorded on a computer running proprietary software.

Data Reduction and Statistical Analyses

Cardiac and metabolic data are presented on a minute-to-minute basis. Predicted heart rates during initial baseline, the stress task, and recovery were computed from the individual regressions of oxygen consumption on heart rate during the last 2 min of the pre exercise rest and the last 2 min of each of the four exercise bouts. The final 2 min were chosen in each case as this would assure a steady state. The 24 regression coefficients were uniformly large (βs ranged from .89 to 1.00, and the mean β, calculated using the z transformation, was .98). By entering oxygen consumption values separately for

each minute of the 8-min baseline, 8-min stress task, and 4-min recovery into the individual regression equations, predicted heart rate values were generated for each of these minutes. A similar strategy was used to calculate predicted cardiac output. Again, the 24 individual oxygen consumption and cardiac output regression coefficients were uniformly large (βs ranged from .87 to 1.00, with the mean coefficient again being .98). Actual and predicted heart rate and cardiac output values were then compared using two factor (actual vs. predicted, time) repeated measures analyses of variance (ANOVAs), applying the Greenhouse–Geisser correction; partial η^2 is reported as a measure of effect size. Finally, average baseline and average stress task values were derived as the mean of the eight individual minute values in each case. Reactivity was then computed as the simple numerical difference between the average task and baseline values. Correlations were computed to examine the association between stress task performance and reactivity for cardiac output and oxygen consumption.

Results

The average minute-by-minute profiles of heart rate, stroke volume, and cardiac output are presented in Figure 1, inspection of which indicates that the stress task substantially increased cardiac output and that increases in both heart rate and stroke volume contributed to this effect. These stress-induced cardiac changes recovered quickly after the end of the stress exposure. The graded sub maximal exercise elicited the expected changes in cardiac output; as the demands of the exercise increased, so too did cardiac output. Although stroke volume increased during exercise, it is clear that heart rate was the main contributor to the orderly rise in cardiac output. The temporal profile of oxygen consumption, presented in Figure 2 is quite different. Whereas during the graded exercise, oxygen consumption increased in the expected orderly fashion, it changed only modestly during the PASAT stress task.

Predicted and actual heart rate and cardiac output during baseline, stress, and recovery are shown in Figure 3.During the 8 min of the stress exposure there is a marked and stable difference between the cardiac activity that was actually recorded and the activity predicted from contemporary levels of oxygen consumption. Indeed, the clarity of the visual evidence for metabolically exaggerated cardiac adjustments during psychological stress is such that statistical tests of difference between predicted and actual cardiac activity seem superfluous. Nevertheless, repeated measures ANOVAs comparing predicted and actual heart rates revealed significant temporal variation, $F(19,437) = 70.27$, $p<.001$, η^2 5.753, a main effect of predicted versus actual heart rate, $F(1,23)536.39$, $p<.001$, $\eta^2 = 613$, and a significant interaction effect, $F(19,437) = 3.60$, $p<.001$, $\eta^2 = 700$; all three effects were large. Even more impressive outcomes emerged from a comparison between predicted and actual cardiac output: for the temporal effect, $F(19,437) = 118.62$, $p<.001$, $\eta^2 = 838$, for predicted versus actual cardiac output, $F(1,23) = 61.24$, $p<.001$, $\eta^2 = 727$, and for the interaction effect, $F(19,437) = 89.92$, $p<.001$, $\eta^2 = 796$.

We have previously found a positive association between performance on the PASAT and cardiac reactivity (Carroll, Phillips, Hunt, & Der, 2007), and, accordingly, correlations were computed between the average change in cardiac output and oxygen consumption during stress exposure on one hand and performance on the PASAT on the other. Whereas change in cardiac output was positively related to performance, $r(22) =.41$, $p =.05$, the association between performance and change in oxygen consumption was negative, $r(22) = -.40$, $p =.05$.

Discussion

There have been few, if any, convincing demonstrations that the functions of the heart may be influenced through pathways that are independent of those which support its linkage to metabolic processes (Brener, 1987, p. 449).

At the time, this could be considered a fair, if somewhat pessimistic, comment. The results of the early human studies examining cardiac activity and energy expenditure in response to acute

psychological stress could at least be regarded as indicative of a dissociation (Carroll, Turner, &Hellawell, 1986; Carroll, Turner, & Prasad, 1986; Langer et al., 1985; Sherwood et al., 1986; Turner & Carroll, 1985). For the past two decades the issue of whether stress elicits metabolically exaggerated cardiac adjustments has been largely neglected, although in the interim substantial research energy has been expended in testing other aspects of the reactivity hypothesis. In addition, a fair deal of attention has been directed at increasing the provocative nature of laboratory stress tasks. In retrospect, the failure to address this fundamental issue is unfortunate, because if increases in cardiac activity in this context are precisely what we would expect on the basis of contemporary energy expenditure, then why might they be hypothesized to be pathophysiological?

We would hope that even the most skeptical commentator would agree that the present results show that a pattern of physiological adjustment to acute psychological stress that is clearly different from that apparent with physical exertion. Whereas for graded exercise increases in heart rate and cardiac output map in an orderly fashion onto increases in oxygen consumption, for psychological stress substantial increases in cardiac activity occur with only the most modest increase in metabolic outlay; that is, the increases in cardiac activity during acute stress exposure would, indeed, appear to be metabolically exaggerated. Comparisons of actual and predicted heart rate and cardiac output reveal substantial differences. Mean (SD) additional heart rate and cardiac output values during the stress task were 17.5 (9.75) bpm and 2.6 (1.13) L/min, with the most reactive participants registering values of 43.0 bpm and 6.1 L/min, respectively; these are not trivial departures from expectations. Thus, our stress task would appear to have elicited metabolically exaggerated cardiac activity of impressive magnitude.

Previous research on additional cardiac output has generally reported that the effect is driven by heart rate with stroke volume changing little during stress exposure (e.g., Carroll et al., 1991; Sherwood et al., 1986). In the present study, significant increases in stroke volume were observed. Three possibilities suggest themselves. First, given that the present participants were, unusually in this sort of research, semi-reclining, there is the issue of posture. Previous research has shown that posture influences the pattern of haemodynamic reactions to stress; testing while seated results in larger cardiac and smaller vascular increases than testing while standing (Sherwood & Turner, 1993; Waldstein, Neumann,& Merrill, 1998). As yet, however, we know of no postural comparisons in this context that has included a semi-reclining condition. Second, the present stress task proved to be somewhat more provocative than those used in earlier studies, where the average increase in cardiac output was around half of that shown here (e.g., Carroll et al., 1991; Sherwood et al., 1986). It is possible that, as with aerobic exercise, stroke volume becomes more implicated in cardiac output changes as the task impact increases. Third, previous studies of additional cardiac output used impedance cardiography, calculating stroke volume from the Kubicek formula (Kubicek et al., 1974). It is possible that the present methodology permits a more sensitive measurement of stroke volume. However, stroke volume derived from impedance cadiography has been observed to correlate highly with stroke volume determined by Doppler echocardiography (Cybulski, Michalak, Kozluk, Piatkowska, & Niewiadomski, 2004; Cybulski, Miskiewicz, Szulc, Torbicki, & Pasierski, 1993; Schmidt et al., 2005). Thus, the stroke volume increases observed in the present study are more likely to reflect posture and task impact than method of measurement.

Recently, researchers have extended the reactivity hypothesis to embrace the profile of cardiovascular recovery from stress exposure (e.g., Brosschot, Pieper, & Thayer, 2005). Indeed, for some, retarded recovery is considered to hold equivalent or even greater implications for pathology than high magnitude reactions per se (Schuler & O'Brien, 1997; Steptoe & Marmot, 2006). In the present study, the substantial stress-related cardiac reactions recovered quickly following stress exposure, and the extent and magnitude of any continuing departure from expectations based on oxygen consumption values were noticeably small. By means of illustration, the mean (SD) additional heart rate and cardiac output values during the 4 min of recovery were 2.0 (4.01) bpm and 0.3 (0.56) L/min, respectively. Clearly, in terms of apparently exaggerated perturbations of cardiac physiology, it is the reactions during stress that catch the eye rather than what happens during recovery. However,

we did not measure blood pressure in this study, and there is evidence that blood pressure recovers much more slowly than cardiac activity (de Boer et al., 2007). Indeed, there would appear to be a shift in the hemodynamic control of blood pressure from early to later stress exposure and recovery, switching from cardiac output to systemic resistance. We have shown that with a more prolonged stress task, a 28-min version of the PASAT, cardiac output returned to baseline by the final quarter of the task and remained at baseline during the recovery period; mean arterial pressure, on the other hand, remained elevated throughout the task and was still above baseline levels 20 min after task completion, as a function of increased resistance (Ring, Burns, & Carroll, 2002).

Whereas the change in oxygen consumption during the stress task was negatively correlated with task performance, the analogous correlation with change in cardiac output was positive. It is possible that the former reflects decreased uncertainty and greater behavioral efficiency among the better performers (Brener, 1987). However, the correlation between performance and cardiac output was positive. Were cardiac output also reflecting only behavioral efficiency, we would have expected a negative correlation. Rather, as has been argued elsewhere (Light, 1981), it would seem that cardiac activity is more readily reflecting mental effort and task engagement. Whatever the case, the pattern of task performance correlations observed in the present study provides further evidence that cardiac adjustments to psychological stress may be influenced by factors other than those that drive metabolic activity.

The study has a number of limitations. First, we tested only men, and, accordingly, our findings cannot be readily generalized to women. Although there is no compelling reason for suspecting that women would not exhibit metabolically exaggerated cardiac reactivity, men and women have been found to differ in their cardiovascular reactions to the present stress task; women show smaller blood pressure, but not heart rate, reactions (Carroll et al., 2007). Second, we tested only healthy participants. It would be informative to extend the study of exaggerated cardiac reactivity to patient populations. We have already reported that young adults with borderline hypertension are more likely to show exaggerated heart rate (Sims & Carroll, 1990) and cardiac output (Carroll et al., 1991) than normotensive controls. However, the phenomenon has yet to be examined in the context of established cardiovascular disease and this would seem an important next step. Third, for reasons cited above, it is unfortunate that we did not measure blood pressure. Such measurement would help clarify the issue of recovery and, along with beat-by-beat heart rate variability, also shed light on the autonomic mechanisms underlying exaggerated cardiac reactivity.

In sum, using a highly provocative psychological stress task and sophisticated, yet noninvasive, methods of measurement of cardiac and metabolic activity, we were able to demonstrate that the cardiac adjustments during psychological stress appear to be substantially exaggerated elative to expectations based on contemporary levels of energy expenditure. Thus, a key axiom of the reactivity hypothesis received strong, if belated, support. The cardiac perturbations during stress were far more impressive in terms of their metabolic inappropriateness than those during recovery. However, it would seem important in future studies of this sort to measure blood pressure, as it recovers more slowly than cardiac activity from stress exposure.

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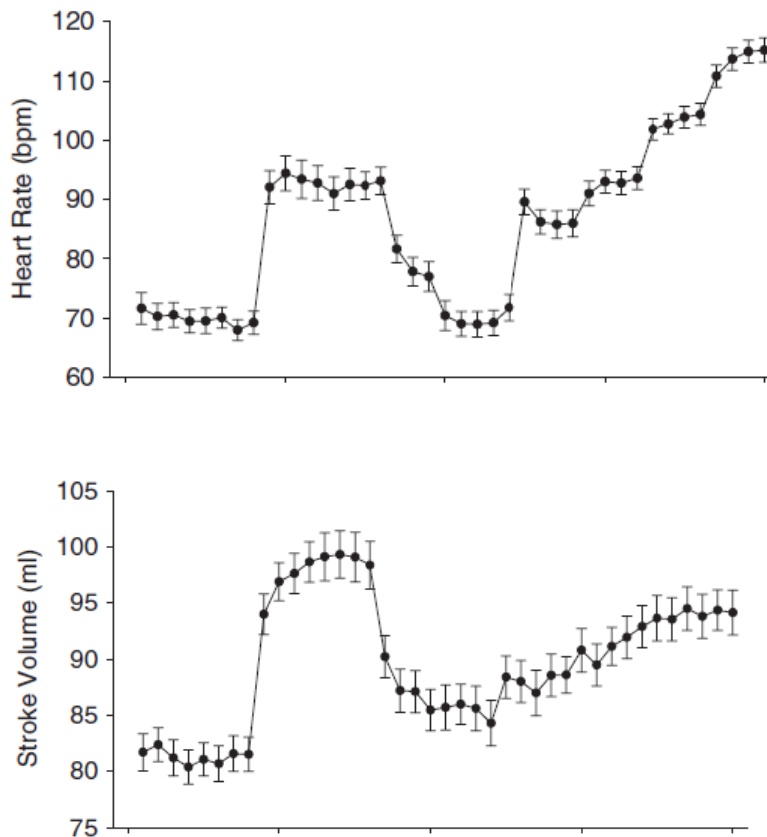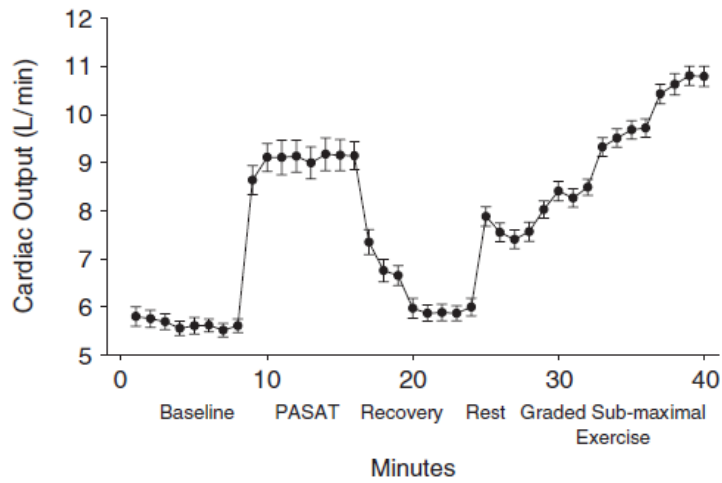
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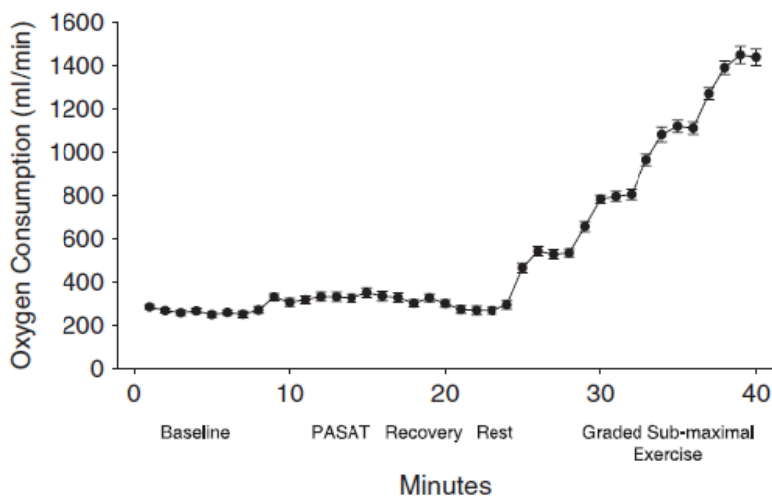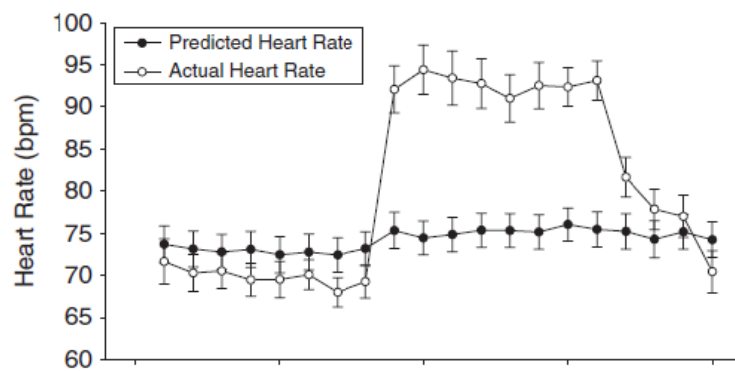Figure 1. The time course of cardiac activity.
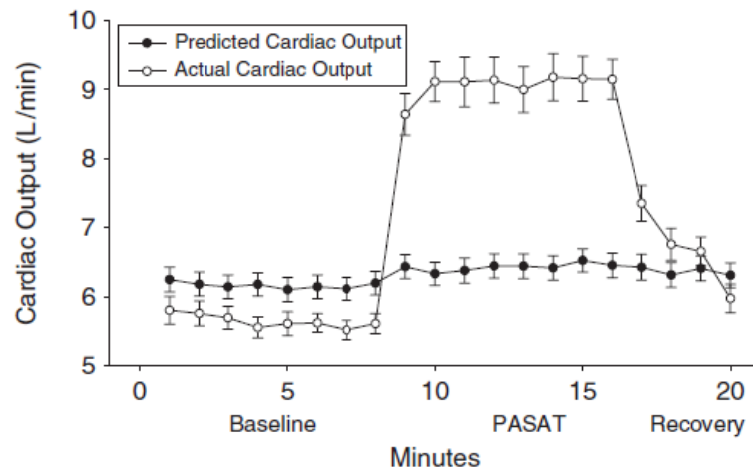

Figure 2. The time course of oxygen consumption.

Figure 3. Predicted versus actual heart rate and cardiac output.